

WEST Search History

DATE: Friday, September 23, 2005

Hide?	Set Name	Query	Hit Count
<i>DB=PGPB; PLUR=YES; OP=ADJ</i>			
<input type="checkbox"/>	L17	L16 not (200?.pray. or 1999.pray. or 1988.pray.)	287
<input type="checkbox"/>	L16	(l13 same l14) and (l13 with l15)	386
<input type="checkbox"/>	L15	bind\$ or family or relat\$	952070
<input type="checkbox"/>	L14	apopto\$	17499
<input type="checkbox"/>	L13	"caspase 8" or mch5 or "mch 5" or mach or flice	5140
<i>DB=EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ</i>			
<input type="checkbox"/>	L12	L11 and l10 and l9	31
<input type="checkbox"/>	L11	bind\$ or family or relat\$	2057329
<input type="checkbox"/>	L10	apopto\$	7210
<input type="checkbox"/>	L9	"caspase 8" or mch5 or "mch 5" or mach or flice	113470
<i>DB=USPT; PLUR=YES; OP=ADJ</i>			
<input type="checkbox"/>	L8	"caspase 8" or mch5 or "mch 5" or mach or flice	11224
<input type="checkbox"/>	L7	L6 not (200?.pray. or 1999.pray. or 1988.pray.)	123
<input type="checkbox"/>	L6	l3 and L5	127
<input type="checkbox"/>	L5	L4 with l1	1266
<input type="checkbox"/>	L4	bind\$ or family or relat\$	3228688
<input type="checkbox"/>	L3	l1 same L2	222
<input type="checkbox"/>	L2	apopto\$	8618
<input type="checkbox"/>	L1	"caspase 8" or mch5 or "mch 5" or mach or flice	11224

END OF SEARCH HISTORY

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DATE: Friday, September 23, 2005

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		<i>DB=EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ</i>	
<input type="checkbox"/>	L12	L11 and l10 and l9	31
<input type="checkbox"/>	L11	bind\$ or family or relat\$	2057329
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<input type="checkbox"/>	L9	"caspase 8" or mch5 or "mch 5" or mach or flice	113470
		<i>DB=USPT; PLUR=YES; OP=ADJ</i>	
<input type="checkbox"/>	L7	L6 not (200?.pray. or 1999.pray. or 1988.pray.)	123
<input type="checkbox"/>	L6	l3 and L5	127
<input type="checkbox"/>	L5	L4 with l1	1266
<input type="checkbox"/>	L4	bind\$ or family or relat\$	3228688
<input type="checkbox"/>	L3	l1 same L2	222
<input type="checkbox"/>	L2	apopto\$	8618
<input type="checkbox"/>	L1	"caspase 8" or mch5 or "mch 5" or mach or flice	11224

END OF SEARCH HISTORY

Set	Items	Description
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	CASPASE(W)(3 OR 8 OR III OR VIII)	
S2	2481373	INHIBIT?
S3	17991	S1 AND S2
S4	229467	APOPTO?
S5	16528	S3 AND S4
S6	2071	S5 NOT PY=2007
S7	1041	S6 NOT PY=1999
S8	1097477	HOMOLOG? OR FAMILY
S9	382	S7 AND S8
S10	168	S9 NOT PY=1998
S11	121	RD (unique items)
S12	17	CASPER AND S4
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	E4	47AU=SCAFFIDI A
	E5	2AU=SCAFFIDI A K
	E6	1AU=SCAFFIDI ABBATE F
	E7	3AU=SCAFFIDI AMELIA
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	E11	22AU=SCAFFIDI C
	E12	1AU=SCAFFIDI C A
	E13	22AU=SCAFFIDI CARSTEN
	E14	1AU=SCAFFIDI CARSTEN A
	E15	2AU=SCAFFIDI D
	E16	1AU=SCAFFIDI D J
	E17	3AU=SCAFFIDI DAVID
	E18	5AU=SCAFFIDI E
	E19	6AU=SCAFFIDI G
	E20	1AU=SCAFFIDI GIORGIO
	E21	1AU=SCAFFIDI IRENE
	E22	8AU=SCAFFIDI JON
	E23	56AU=SCAFFIDI L
	11/6/1	(Item 1 from file: 155) 12391635 PMID: 9441900 Effects of redox-related congeners of NO on apoptosis and caspase - 3 activity. Aug 1997
	11/6/2	(Item 2 from file: 155) 12127798 PMID: 9430228 TRAIL receptors 1 (DR4) and 2 (DR5) signal FADD -dependent apoptosis and activate NF-kappaB. Dec 1997
	11/6/3	(Item 3 from file: 155) 12120892 PMID: 9420624 Involvement of ICE (Caspase) family in gamma-radiation-induced apoptosis of normal B lymphocytes. Dec 1997
	11/6/4	(Item 4 from file: 155) 12111893 PMID: 9409814 Differential activity of bcl-2 and ICE enzyme: family protease inhibitors on Fas and puromycin-induced apoptosis of glioma cells. Nov 27 1997
	11/6/5	(Item 5 from file: 15) 12108221 PMID: 9406913 Activation of CPP-32 protease in hippocampal neurons following ischemia and epilepsy. Oct 15 1997
	E24	1AU=SCAFFIDI L E
	E25	1AU=SCAFFIDI L F
	E26	10AU=SCAFFIDI M
	E27	3AU=SCAFFIDI M G
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	E29	1AU=SCAFFIDI MARIA G
	E30	1AU=SCAFFIDI MARIAGRAZIA
	E31	2AU=SCAFFIDI MARIELLA
	E32	4AU=SCAFFIDI P
	E33	18AU=SCAFFIDI PAOLA
	E34	2AU=SCAFFIDI ROBERT C
	E35	2AU=SCAFFIDI S
	E36	64AU=SCAFFIDI V
	E37	1AU=SCAFFIDI AMELIA K
	E38	2AU=SCAFFINO L
	E39	1AU=SCAFFINO M
	S13	47 E3, E11-E14
	S14	44 S4 AND S13
	S15	26 S2 AND S14
	S16	2 AU=PETER'
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	E4	16AU=PETER M A
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	E13	4AU=PETER M I
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	S19	57 S18 AND S2
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	E36	2AU=KRAMER PAUL R JR
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	E48	5AU=KRAMER PETRA M
	S23	1048 E3-E24, E42-E47
	S24	3 S23 AND (S2 AND S4)NOT (S20 OR S15)
	Baculovirus inhibitors of apoptosis (IAPs) block activation of Sf-caspase-1. Dec 9 1997	
	11/6/11	(Item 11 from file: 15) 12090599 PMID: 9388494 Nitric oxide reversibly inhibits seven members of the caspase family via S-nitrosylation. Nov 17 1997
	11/6/12	(Item 12 from file: 15) 12087561 PMID: 9384571 The c-IAP-1 and c-IAP-2 proteins are direct inhibitors of specific caspases. Dec 1 1997
	11/6/13	(Item 13 from file: 15) 12085478 PMID: 9374516 Cell-specific induction of apoptosis by microinjection of cytochrome c. Bcl-xL has activity independent of cytochrome c release. Nov 28 1997
	11/6/14	(Item 14 from file: 15) 12080169 PMID: 9368003 Specific proteolysis of the kinase protein kinase C-related kinase 2 by caspase - 3 during apoptosis. Identification by a novel, small pool expression cloning strategy. Nov 21 1997
	11/6/15	(Item 15 from file: 15) 12074622 PMID: 9362518

Fas induces cytoplasmic apoptotic responses and activation of the MKK7-JNK/SAPK and MKK6-p38 pathways independent of CPP32-like proteases. Nov 17 1997

11/6/16 (Item 16 from file: 15 12069881 PMID: 9352442)

Caspase-dependent apoptosis of COS-7 cells induced by Bax overexpression: differential effects of Bcl-2 and Bcl-xL on Bax-induced caspase activation and apoptosis. Oct 9 1997

11/6/17 (Item 17 from file: 15 12069062 PMID: 9376593)

Cross resistance of CD95- and drug-induced apoptosis as a consequence of deficient activation of caspases (ICE/Ced-3 proteases). Oct 15 1997

11/6/18 (Item 18 from file: 15 12064799 PMID: 9380701)

CLARP, a death effector domain-containing protein interacts with caspase - 8 and regulates apoptosis. Sep 30 1997

11/6/19 (Item 19 from file: 15 12062291 PMID: 9357850)

Involvement of the ICE family of proteases in silica-induced apoptosis in human alveolar macrophages. Oct 1997

11/6/20 (Item 20 from file: 15 12058266 PMID: 9354463)

Benfonic acid triggers CD95 (APO-1/Fas) and p53-independent apoptosis via activation of caspases in neuroectodermal tumors. Nov 1 1997

11/6/21 (Item 21 from file: 15 12055928 PMID: 9348308)

Specific activation of the cysteine protease CPP32 during the negative selection of T cells in the thymus. Nov 3 1997

11/6/22 (Item 22 from file: 15 12055292 PMID: 9350436)

Molecular mechanisms of apoptosis in HL-60 cells induced by a nitric oxide-releasing compound. Sep 1997

11/6/23 (Item 23 from file: 15 12051929 PMID: 8987778)

Activation of the CED3/ICE-related protease CPP32 in cerebellar granule neurons undergoing apoptosis but not necrosis. Jan 15 1997

11/6/24 (Item 24 from file: 15 12049903 PMID: 9342343)

Activation of distinct caspase-like proteases by Fas and reaper in *Drosophila* cells. Oct 28 1997

11/6/25 (Item 25 from file: 15 12047583 PMID: 9343438)

A functional role for death proteases in s-Myc- and c-Myc-mediated apoptosis. Nov 1997

11/6/26 (Item 26 from file: 15 12047414 PMID: 9343261)

Bovine herpesvirus 4 BORF2 protein inhibits Fas- and tumor necrosis factor receptor 1-induced apoptosis and contains death effector domains shared with other gamma-2 herpesviruses. Nov 1997

11/6/27 (Item 27 from file: 15 12033737 PMID: 9325343)

Cleavage of focal adhesion kinase by caspases during apoptosis. Oct 10 1997

11/6/28 (Item 28 from file: 15 12033642 PMID: 9325248)

Identification and molecular cloning of two novel receptors for the cytotoxic ligand TRAIL. Oct 10 1997

11/6/29 (Item 29 from file: 15 12032487 PMID: 9323209)

Caspase - 3 - generated fragment of gelsolin: effector of morphological change in apoptosis. Oct 10 1997

11/6/30 (Item 30 from file: 15 12031147 PMID: 9324287)

Inhibition of caspase proteases by CrmA enhances the resistance of human leukemic cells to multiple chemotherapeutic agents. Oct 1997

11/6/31 (Item 31 from file: 15 12028417 PMID: 9314559)

Activation of CPP32-like proteases is not sufficient to trigger apoptosis: inhibition of apoptosis by agents that suppress activation of AP24, but not CPP32-like activity. Oct 6 1997

11/6/32 (Item 32 from file: 15 12028398 PMID: 9314540)

Bax deletion further orders the cell death pathway in cerebellar granule cells and suggests a caspase-independent pathway to cell death. Oct 6 1997

11/6/33 (Item 33 from file: 15 12026778 PMID: 9311998)

TRAIL-R2: a novel apoptosis -mediating receptor for TRAIL. Sep 1 1997

11/6/34 (Item 34 from file: 15 12023747 PMID: 9311830)

The ability of BHRF1 to inhibit apoptosis is dependent on stimulus and cell type. Oct 1997

11/6/35 (Item 35 from file: 15 12015818 PMID: 9303309)

Inhibition of apoptosis by the actin-regulatory protein gelsolin. Aug 1 1997

11/6/36 (Item 36 from file: 15 12013839 PMID: 9300088)

Fluorometric and colorimetric detection of caspase activity associated with apoptosis. Aug 15 1997

11/6/37 (Item 37 from file: 15 12004278 PMID: 9287312)

Requirement of the caspase - 3 CPP32 protease cascade for apoptotic death following cytokine deprivation in hematopoietic cells. Sep 12 1997

11/6/38 (Item 38 from file: 15 11955875 PMID: 9289491)

CASH, a novel caspase homologue with death effector domains. Aug 8 1997

11/6/39 (Item 39 from file: 15 11979278 PMID: 9261102)

Activation of caspase-2 in apoptosis. Aug 22 1997

11/6/40 (Item 40 from file: 15 11979028 PMID: 9264376)

Camplotheclin-induced apoptosis in p53-null human leukemia HL60 cells and their isolated nuclei: effects of the protease inhibitors Z-VAD-fmk and dichloroisocoumarin suggest an involvement of both caspases and serine proteases. Aug 1997

11/6/41 (Item 41 from file: 15 11972119 PMID: 9257710)

CPP32 activation during dolchylphosphate-induced apoptosis in U937 leukemia cells. Jul 21 1997

11/6/42 (Item 42 from file: 15 11955894 PMID: 9236228)

Establishment of a cell-free system of neuronal apoptosis: comparison of premitochondrial, mitochondrial, and postmitochondrial phases. Aug 5 1997

11/6/43 (Item 43 from file: 15 11956118 PMID: 9240442)

Nitric oxide inhibits CPP32-like activity under redox regulation. Jul 18 1997

11/6/44 (Item 44 from file: 15 11955986 PMID: 9235961)

The large subunit of the DNA replication complex C (DSEBRF-C140) cleaved and inactivated by caspase - 3 (CPP32/YAMA) during Fas-induced apoptosis. Aug 1 1997

11/6/45 (Item 45 from file: 15 11951429 PMID: 9233763)

Involvement of caspase-4(-like) protease in Fas-mediated apoptotic pathway. Jul 17 1997

11/6/46 (Item 46 from file: 15 11948649 PMID: 9230442)

X-linked IAP is a direct inhibitor of cell-death proteases. Jul 17 1997

11/6/47 (Item 47 from file: 15 11948244 PMID: 9228018)

FLAME-1, a novel FADD-like anti-apoptotic molecule that regulates Fas/TNFR1-induced apoptosis. Jul 25 1997

11/6/48 (Item 48 from file: 15 11940223 PMID: 9219695)

Alternative cleavage of Alzheimer-associated presenilins during apoptosis by a caspase - 3 family protease. Jul 18 1997

11/6/49 (Item 49 from file: 15 11938036 PMID: 9218876)

Involvement of caspase family proteases in transforming growth factor-beta-induced apoptosis. Jul 1997

11/6/50 (Item 50 from file: 15 11935875 PMID: 9217161)

Inhibition of death receptor signals by cellular FLIP. Jul 10 1997

11/6/51 (Item 51 from file: 15 11928659 PMID: 9208847)

Casper is a FADD - and caspase-related inducer of apoptosis. Jun 1997

11/6/52 (Item 52 from file: 15 11923011 PMID: 9202418)

The apoptotic cysteine protease CPP32. Mar 1997

11/6/53 (Item 53 from file: 15 11921069 PMID: 9200447)

Bcl-2 expression in target cells leads to functional inhibition of caspase - 3 protease family in human NK and lymphokine-activated killer cell granule-mediated apoptosis. Jul 1 1997

11/6/54 (Item 54 from file: 15 11914983 PMID: 9194224)

Do CTL kill target cells by inducing apoptosis? Apr 1997

11/6/55 (Item 55 from file: 15 11890620 PMID: 916641)

Temporal phases in apoptosis defined by the actions of Src homology 2 domains, ceramide, Bcl-2, interleukin-1beta converting enzyme family proteases, and a dense membrane fraction. Jun 2 1997

11/6/56 (Item 56 from file: 15 11889223 PMID: 9166725)

Characterization of CPP32-like protease activity following apoptotic challenge in SH-SY5Y neuroblastoma cells. Jun 1997

11/6/57 (Item 57 from file: 15 11882268 PMID: 9157970)

Intracellular ATP levels determine cell death fate by apoptosis or necrosis. May 15 1997

11/6/58 (Item 58 from file: 15 11871241 PMID: 9144536)

Specific cleavage of the large subunit of replication factor C in apoptosis is mediated by CPP32-like protease. Apr 17 1997

11/6/59 (Item 59 from file: 15 11860478 PMID: 9115219)

Cleavage of PITSRE kinases by ICE/CASP-1 and CPP32/CASP-3 during apoptosis induced by tumor necrosis factor. May 2 1997

11/6/60 (Item 60 from file: 15 11859622 PMID: 9130145)

Activation of CPP32 during apoptosis of neurons and astrocytes. Apr 15 1997

11/6/61 (Item 61 from file: 15 11851386 PMID: 9112440)

Essential role of active nuclear transport in apoptosis. Jan 1997

11/6/62 (Item 62 from file: 15 11844298 PMID: 9104814)

TRAF-interacting protein (TRIP): a novel component of the tumor necrosis factor receptor (TNFR) and CD30-TRAF signaling complexes that inhibits TRAF2-mediated NF-kappaB activation. Apr 7 1997

11/6/63 (Item 63 from file: 15 11835766 PMID: 9092497)

Substrate specificities of caspase family proteases. Apr 11 1997

11/6/64 (Item 64 from file: 15 11835757 PMID: 9092488)

A novel family of viral death effector domain-containing molecules that inhibit both CD-95- and tumor necrosis factor receptor-1-induced apoptosis. Apr 11 1997

11/6/65 (Item 65 from file: 15 11830217 PMID: 9087414)

Viral FLICE-inhibitory proteins (FLIPs) prevent apoptosis induced by death receptors. Apr 3 1997

11/6/66 (Item 66 from file: 15 11815610 PMID: 9070890)

Specific expression of CPP32 in sensory neurons of mouse embryos and activation of CPP32 in the apoptosis induced by a withdrawal of NGF. Feb 24 1997

11/6/67 (Item 67 from file: 15 11815499 PMID: 9069264)

Transducing signals of life and death. Apr 1997

11/6/68 (Item 68 from file: 15 11815330 PMID: 9070648)

Actin cleavage by CPP-32/appain during the development of apoptosis. Mar 6 1997

11/6/69 (Item 69 from file: 15 11810527 PMID: 9065443)

Target protease specificity of the viral serpin CrmA. Analysis of five caspases. Mar 21 1997

11/6/70 (Item 70 from file: 15 11805660 PMID: 9058785)

Apoptosis of immature thymocytes mediated by E2/CD99. Mar 15 1997

11/6/71 (Item 71 from file: 15 11801838 PMID: 9054391)

Selective activation of caspases during apoptotic induction in HL-60 cells. Effects of a tetrapeptide inhibitor. Mar 14 1997

11/6/72 (Item 72 from file: 15 11798789 PMID: 9050895)

Inhibition of interleukin 1beta converting enzyme family proteases reduces ischemic and excitotoxic neuronal damage. Mar 4 1997

11/6/73 (Item 73 from file: 15 11795229 PMID: 9045686)

- Fas-associated death domain protein interleukin-1 β -converting enzyme 2 (FLICE2), an ICE/ Ced-3 homologue, is proximally involved in CD95- and p55-mediated death signaling. Mar 7 1997
- 11/6/74 (Item 74 from file: 15 11789639 PMID: 9038883 Development and initial application of an in vitro model of apoptosis in rodent cholangiocytes. Jan 1997
- 11/6/75 (Item 75 from file: 15 11788223 PMID: 9037206 The *Caeonchaditis* elegans death protein Ced-4 contains a motif with similarity to the mammalian 'death effector domain'. Feb 3 1997
- 11/6/76 (Item 76 from file: 15 11788048 PMID: 9037025 Death effector domain-containing herpesvirus and poxvirus proteins inhibit both Fas- and TNFR1-induced apoptosis. Feb 18 1997
- 11/6/77 (Item 77 from file: 15 11787967 PMID: 9036942 Inhibition of CPP32-like proteases prevents granzyme B- and Fas-, but not granzyme A-based cytotoxicity exerted by CTL clones. Mar 1 1997
- 11/6/78 (Item 78 from file: 15 11782973 PMID: 9027312 Interaction of CED-4 with CED-3 and CED-9: a molecular framework for cell death. Feb 21 1997
- 11/6/79 (Item 79 from file: 15 11779909 PMID: 9023346 Activation of the cell death program by inhibition of proteasome function. Feb 4 1997
- 11/6/80 (Item 80 from file: 15 11771863 PMID: 9006941 FLICE induced apoptosis in a cell-free system. Cleavage of caspase zymogens. Jan 31 1997
- 11/6/81 (Item 81 from file: 15 11769092 PMID: 9001220 Characterization of reaper- and FADD -induced apoptosis in a lepidopteran cell line. Feb 1997
- 11/6/82 (Item 82 from file: 15 11764419 PMID: 8998038 Baculovirus P35 inhibits the glucocorticoid-mediated pathway of cell death. Jan 1 1997
- 11/6/83 (Item 83 from file: 15 11762692 PMID: 8977187 IL-1 β convertase (ICE) does not play a requisite role in apoptosis induced in T lymphoblasts by Fas-dependent or Fas-independent CTL effector mechanisms. Jan 1 1997
- 11/6/84 (Item 84 from file: 15 11760463 PMID: 14646542 Caspase -3 /CPP32-like activity is not sufficient to mediate apoptosis in an IL-2 dependent T cell line. 1997
- 11/6/85 (Item 85 from file: 15 11638061 PMID: 8947555 Genetic and metabolic status of NGF-deprived sympathetic neurons saved by an inhibitor of ICE family proteases. Dec 1996
- 11/6/86 (Item 86 from file: 15 11631522 PMID: 8943013 Inhibition of interleukin 1 β -converting enzyme-mediated apoptosis of mammalian cells by baculovirus IAP. Nov 26 1996
- 11/6/87 (Item 87 from file: 15 11627887 PMID: 8940042 Induction of CPP32-like activity in PC12 cells by withdrawal of trophic support. Dissociation from apoptosis. Nov 29 1996
- 11/6/88 (Item 88 from file: 15 11603063 PMID: 8912630 Fas-mediated stimulation induces IL-8 secretion by rheumatoid arthritis synoviocytes independently of CPP32-mediated apoptosis. Nov 1 1996
- 11/6/89 (Item 89 from file: 15 11597660 PMID: 8906799 Protection against Fas/APO-1- and tumor necrosis factor-mediated cell death by a novel protein, sentrin. Nov 15 1996
- 11/6/106 (Item 106 from file: 15 11336603 PMID: 8642305 Apoptain/CPP32 cleaves proteins that are essential for cellular repair: a fundamental principle of apoptotic death. May 1 1996
- 11/6/107 (Item 107 from file: 15 11329727 PMID: 8647264 Ligation of CD40 rescues Ramos-Burkitt lymphoma B cells from calcium ionophore- and antigen receptor-triggered apoptosis by inhibiting activation of the cysteine protease CPP32/Yama and cleavage of its substrate PARP. May 20 1996
- 11/6/108 (Item 108 from file: 15 11323076 PMID: 8665848 The cytotoxic cell protease granzyme B initiates apoptosis in a cell-free system by proteolytic processing and activation of the ICE/CED-3 family protease, CPP32, via a novel two-step mechanism. May 15 1996
- 11/6/109 (Item 109 from file: 15 11314298 PMID: 8626669 DA-GDI, a substrate of CPP32, is proteolyzed during Fas-induced apoptosis. May 10 1996
- 11/6/110 (Item 110 from file: 15 11312002 PMID: 8643514 Cloning and expression of apoptosis inhibitory protein homologs that function to inhibit apoptosis and/or bind tumor necrosis factor receptor-associated factors. May 14 1996
- 11/6/111 (Item 111 from file: 15 11305508 PMID: 8614469 Sequential activation of ICE-like and CPP32-like proteases during Fas-mediated apoptosis. Apr 25 1996
- 11/6/112 (Item 112 from file: 15 11294953 PMID: 8619857 Bcl-2 overexpression blocks activation of the death protease CPP32/Yama/apoptain. Apr 16 1996
- 11/6/113 (Item 113 from file: 15 11287993 PMID: 8620480 Involvement of CPP32/Yama(-like) proteases in Fas-mediated apoptosis. Apr 15 1996
- 11/6/114 (Item 114 from file: 15 11253242 PMID: 8567626 CPP32/apoptain is a key interleukin 1 β converting enzyme-like protease involved in Fas-mediated apoptosis. Jan 26 1996
- 11/6/115 (Item 115 from file: 15 11234430 PMID: 8548810 The TNFR2-TRAF signaling complex contains two novel proteins related to baculoviral inhibitor of apoptosis proteins. Dec 29 1995
- 11/6/116 (Item 116 from file: 15 10993263 PMID: 7774019 Yama/CPP32 beta, a mammalian homolog of CED-3, is a Cma-A inhibitable protease that cleaves the death substrate poly(ADP-ribose) polymerase. Jun 2 1995
- 11/6/117 (Item 117 from file: 15 10979123 PMID: 7538907 FADD, a novel death domain-containing protein, interacts with the death domain of Fas and initiates apoptosis. May 19 1995
- 11/6/118 (Item 118 from file: 0011413824 BIOSIS NO.: 199800208071 Bcl-2 prevents TNF- and Fas-induced cell death but does not inhibit initial processing of caspase-3 1997
- 11/6/119 (Item 119 from file: 0011292489 BIOSIS NO.: 199800086736 Disulfiram in a potent inhibitor of proteases of the caspase family 1997
- 11/6/120 (Item 120 from file: 0011241387 BIOSIS NO.: 199800035634 Ligation of CD40 potentiates Fas-mediated activation of the cysteine protease CPP32, cleavage of its death substrate PARP, and apoptosis in Ramos-Burkitt lymphoma B cells 1997
- 11/6/121 (Item 121 from file: 0011163416 BIOSIS NO.: 199799797476 Caspase-dependent apoptosis of COS-7 cells induced by Bax overexpression: Differential effects of Bcl-2 and Bcl-x-L on Bax-induced caspase activation and apoptosis 1997
- Enzyme No.: EC 3.4.22. (Cysteine Endopeptidases); EC 3.4.22.- (caspase -3); EC 3.4.22.36 (Caspase 1)
- 11/7/12 (Item 12 from file: 15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv. 12087561 PMID: 9384571 The c-IAP-1 and c-IAP-2 proteins are direct inhibitors of specific caspases. Roy N; Devereaux Q L; Takahashi R; Salvesen G S; Reed J C The Burnham Institute, Program on Apoptosis and Cell Death Research, 10901 North Torrey Pines Road, La Jolla, CA 92037, USA. EMBO journal (ENGLAND) Dec 1 1997, 16 (23) p6914-25, ISSN 0261-4189 Journal Code: 8208664

Contract/Grant No.: CA-69381; CA; NCI Publishing Model Print Document type: Journal Article

Languages: ENGLISH Main Citation Owner: NLM Record type: MEDLINE; Completed

The inhibitor of apoptosis (IAP) family of proteins are highly conserved through evolution. However, the mechanisms by which these proteins interfere with apoptotic cell death have been enigmatic. Recently, we showed that one of the human IAP family proteins, XIAP, can bind to and potentially inhibit specific cell death proteases (caspases) that function in the distal portions of the proteolytic cascades involved in apoptosis. In this study, we investigated three of the other known members of the human IAP family, c-IAP-1, c-IAP-2 and NAIP. Similarly to XIAP, in vitro binding experiments indicated that c-IAP-1 and c-IAP-2 bound specifically to the terminal effector cell death proteases, caspases-3 and -7, but not to the proximal protease caspase -8, caspases-1 or -6. In contrast, NAIP failed to bind tightly to any of these proteases. Recombinant c-IAP-1 and c-IAP-2 also inhibited the activity of caspases-3 and -7 in vitro, with estimated K_{is} of ≤ 0.1 μ M, whereas NAIP did not. The BIR domain-containing region of c-IAP-1 and c-IAP-2 was sufficient for inhibition of these caspases, though proteins that retained the RING domain were somewhat more potent. Utilizing a cell-free system in which caspases were activated in cytosolic extracts by addition of cytochrome c, c-IAP-1 and c-IAP-2 inhibited both the generation of caspase activities and proteolytic processing of procaspase -3. Similar results were obtained in intact cells when c-IAP-1 and c-IAP-2 were overexpressed by gene transfection, and apoptosis was induced by the anticancer drug, etoposide. Cleavage of c-IAP-1 or c-IAP-2 was not observed when interacting with the caspases, implying a different mechanism from the baculovirus p35 protein, the broad spectrum suicide inactivator of caspases. Taken together, these findings suggest that c-IAP-1 and c-IAP-2 function similarly to XIAP by inhibiting the distal cell death proteases, caspases-3 and -7, whereas NAIP presumably inhibits apoptosis via other targets.

Record Date Created: 19980129 Record Date Completed: 19980129

11/5/38 (Item 38 from file: 15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.

11958675 PMID: 9289491

CASH, a novel caspase homologue with death effector domains.

Goltsev Y V, Kovalenko A V, Arnold E, Varfolomeev E E, Brodianski V M, Wallach D

Department of Membrane Research and Biophysics, Weizmann Institute of Science, 76100 Rehovot, Israel.

Journal of biological chemistry (UNITED STATES) Aug 8 1997, 272 (32) p19641-4, ISSN 0021-9258 Journal Code: 2985121R

Publishing Model Print Document type: Journal Article Languages: ENGLISH

Main Citation Owner: NLM Record type: MEDLINE; Completed Subfile: INDEX MEDICUS

CASP-8 and CASP-10, members of a cysteine protease family that participates in apoptosis, interact with MORT1/FADD, an adapter protein in the CD120a (p55 tumor necrosis factor receptor), and CD95 (Fas/Apo-1) death-inducing signaling pathways, through a shared N-terminal sequence motif, the death effector domain. We report cloning of two splice variants of a novel protein, CASH, that contain two N-terminal death effector domains and can bind through them to each other, to MORT1/FADD, to CASP-8, and to CASP-10. The unique C-terminal part of the longer variant shows marked sequence homology to the caspase protease region yet lacks several of the conserved caspase active site residues, suggesting that it is devoid of cysteine protease activity. Overexpression of the short CASH splice variant strongly inhibited cytotoxicity induction by CD120a and CD95. Expression of the longer variant, while inhibiting cytotoxicity in HeLa cells, had a marked cytotoxic effect in 293 cells that could be shown to involve its protease homology region. The findings suggest that CASH acts as an attenuator and/or initiator in CD95 and CD120a signaling for cell death.

Tags: Research Support, Non-U.S. Gov't

Descriptors: *Adaptor Proteins, Signal Transducing; *Apoptosis; *Caspases; *Cysteine Endopeptidases--chemistry--CH; *Intracellular Signaling Peptides and Proteins; Amino Acid Sequence; Animals; Antigens, CD; --metabolism--ME; Antigens, CD95--metabolism--ME; Carrier Proteins--chemistry--CH; Carrier Proteins--metabolism--ME; Caspase 1; Cloning, Molecular; Cysteine Endopeptidases--metabolism--ME; Hela Cells; Humans; Interleukin-1--metabolism--ME; Mice; Molecular Sequence Data; Receptors; Tumor Necrosis Factor--metabolism--ME; Receptors, Tumor Necrosis Factor, Type 1; Saccharomyces cerevisiae; Sequence Homology, Amino Acid; Transfection Molecular Sequence Databank No.: GENBANK/Y14039; GENBANK/Y14040; GENBANK/Y14041; GENBANK/Y14042

CAS Registry No.: 0 (Adaptor Proteins, Signal Transducing); 0 (Antigens, CD); 0 (Antigens, CD95); 0 (CASP8 and FADD-like apoptosis regulating protein); 0 (Carrier Proteins); 0 (Interleukin-1); 0 (Intracellular Signaling Peptides and Proteins); 0 (MORT1 protein); 0 (Receptors, Tumor Necrosis Factor); 0 (Receptors, Tumor Necrosis Factor, Type 1)

Enzyme No.: EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.22.- (Caspases); EC 3.4.22.- (interleukin 1beta-converting enzyme 2); EC 3.4.22.36 (Caspase 1) Record Date Created: 19970905 Record Date Completed: 19970905

11/5/46 (Item 46 from file: 15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.

11948649 PMID: 9230442

X-linked IAP is a direct inhibitor of cell-death proteases.

Deveraux Q L, Takahashi R, Salvesen G S, Reed J C

The Burnham Institute, Program on Apoptosis and Cell Death Research, La Jolla, California 92037, USA.

Nature (ENGLAND) Jul 10 1997, 388 (6639) p300-4, ISSN 0028-0836 Journal Code: 0410462

Publishing Model Print Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM

Record type: MEDLINE; Completed Subfile: INDEX MEDICUS

The inhibitor -of- apoptosis (IAP) family of genes has an evolutionarily conserved role in regulating programmed cell death in animals ranging from insects to humans. Ectopic expression of human IAP proteins can suppress cell death induced by a variety of stimuli, but the mechanism of this inhibition was previously unknown. Here we show that human X-chromosome-linked IAP directly inhibits at least two members of the caspase family of cell-death proteases, caspase -3 and caspase-7. As the caspases

are highly conserved throughout the animal kingdom and are the principal effectors of apoptosis, our findings suggest how IAPs might inhibit cell death, providing evidence for a mechanism of action for these mammalian cell-death suppressors.

Descriptors: *Apoptosis--physiology--PH; *Caspases; *Cysteine Endopeptidases--metabolism--ME; *Cysteine Proteinase Inhibitors--physiology--PH; *Protein--physiology--PH; *Proto-Oncogene Proteins c-bcl-2; *X Chromosome; Amino Acid Sequence; Caspase 1; Cell Nucleus--metabolism--ME; Cell-Free System; Cytochrome c Group--metabolism--ME; Cytosol--metabolism--ME; Enzyme Activation; Humans; Jurkat Cells; Linkage (Genetics); Molecular Sequence Data; Protein Processing, Post-Translational; Proteins--genetics--GE; Proto-Oncogene Proteins--metabolism--ME; Recombinant Proteins--metabolism--ME; Transfection Molecular Sequence Databank No.: GENBANK/U32974

CAS Registry No.: 0 (Bax protein); 0 (Cysteine Proteinase Inhibitors); 0 (Cytochrome c Group); 0 (IAP-like protein, vertebrate); 0 (Proteins); 0 (Proto-Oncogene Proteins); 0 (Proto-Oncogene Proteins c-bcl-2); 0 (Recombinant Proteins)

Enzyme No.: EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.22.- (Caspases); EC 3.4.22.- (caspase 7); EC 3.4.22.- (caspase -3); EC 3.4.22.36 (Caspase 1) Record Date Created: 19970807 Record Date Completed: 19970807

11/5/47 (Item 47 from file: 15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.

11948244 PMID: 9228018

FLAME-1, a novel FADD-like anti-apoptotic molecule that regulates Fas/TNFR1-induced apoptosis.

Srinivasula S M, Ahmad M, Otilili S, Bullrich F, Banks S, Wang Y, Fernandes-Alnemri T, Croce C M, Litwack G, Tomaselli K J, Armstrong R C, Alnemri E S

Center for Apoptosis Research and the Department of Microbiology and Immunology, Kimmel Cancer Institute, Thomas Jefferson University, Philadelphia, Pennsylvania 19107, USA.

Journal of biological chemistry (UNITED STATES) Jul 25 1997, 272 (30) p18542-5, ISSN 0021-9258 Journal Code: 2985121R

Contract/Grant No.: AG 13487, AG, NIA Publishing Model Print Document type: Journal Article

Languages: ENGLISH Main Citation Owner: NLM Record type: MEDLINE; Completed Subfile: INDEX MEDICUS

We identified and cloned a novel human protein that contains FADD/Mor1 death effector domain homology regions, designated FLAME-1. FLAME-1, although most similar in structure to Mch4 and Mch5, does not possess caspase activity but can interact specifically with FADD, Mch4, and Mch5. Interestingly, FLAME-1 is recruited to the Fas receptor complex and can abrogate Fas/TNFR-induced apoptosis upon expression in Fas/tumor necrosis factor-sensitive MCF-7 cells, possibly by acting as a dominant-negative inhibitor. These findings identify a novel endogenous control point that regulates Fas/TNFR1-mediated apoptosis.

Tags: Research Support, U.S. Gov't, P.H.S.

Descriptors: *Adaptor Proteins, Signal Transducing; *Apoptosis; *Carrier Proteins--metabolism--ME; *Caspases; *Intracellular Signaling Peptides and Proteins; *Proteins--metabolism--ME; Amino Acid Sequence; Apoptosis--drug effects--DE; Apoptosis--radiation effects--RE; Carrier Proteins--chemistry--CH; Carrier Proteins--genetics--GE; Cloning, Molecular; Cysteine Endopeptidases--chemistry--CH; Cysteine Endopeptidases--metabolism--ME; Humans; Membrane Glycoproteins--metabolism--ME; Molecular Sequence Data; Sequence Homology, Amino Acid; TNF Receptor-Associated Factor 1; Tissue Distribution; Ultraviolet Rays Molecular Sequence Databank No.: GENBANK/AF009616; GENBANK/AF009617; GENBANK/AF009618; GENBANK/AF009619; GENBANK/AF009620

CAS Registry No.: 0 (Adaptor Proteins, Signal Transducing); 0 (CASP8 and FADD-like apoptosis regulating protein); 0 (Carrier Proteins); 0 (FasL protein); 0 (Intracellular Signaling Peptides and Proteins); 0 (MORT1 protein); 0 (Membrane Glycoproteins); 0 (Proteins); 0 (TNF Receptor-Associated Factor 1)

Enzyme No.: EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.22.- (Caspases); EC 3.4.22.- (caspase 10); EC 3.4.22.- (caspase 8); EC 3.4.22.- (caspase 9) Record Date Created: 19970909 Record Date Completed: 19970909

11/5/50 (Item 50 from file: 15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.

11935875 PMID: 9217161

Inhibition of death receptor signals by cellular FLIP.

Imler M, Thome M, Hahne M, Schneider P, Hofmann K, Steiner J L, Schroter M, Burns K, Mattmann C, Rimoldi D, French L E, Tschoop J

Institute of Biochemistry, Lausanne branch, University of Lausanne, Switzerland.

Nature (ENGLAND) Jul 10 1997, 388 (6638) p190-5, ISSN 0028-0836 Journal Code: 0410462

Publishing Model Print; Comment in Nature. 1997 Jul 10;388(6638) 123, 125-6; Comment in PMID 9217148

Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: MEDLINE; Completed Subfile: INDEX MEDICUS

The widely expressed protein Fas is a member of the tumour necrosis factor receptor family which can trigger apoptosis. However, Fas surface expression does not necessarily render cells susceptible to Fas ligand-induced death signals, indicating that inhibitors of the apoptosis-signalling pathway must exist. Here we report the characterization of an inhibitor of apoptosis, designated FLIP (for FLICE-inhibitory protein), which is predominantly expressed in muscle and lymphoid tissues. The short form, FLIPs, contains two death effector domains and is structurally related to the viral FLIP inhibitors of apoptosis, whereas the long form, FLIP(L), contains in addition a caspase-like domain in which the active-centre cysteine residue is substituted by a tyrosine residue. FLIPs and FLIP(L) interact with the adaptor protein FADD and the protease FLICE, and potentially inhibit apoptosis induced by all known human death receptors. FLIP(L) is expressed during the early stage of T-cell activation, but disappears when T cells become susceptible to Fas ligand-mediated apoptosis. High levels of FLIP(L) protein are also detectable in melanoma cell lines and malignant melanoma tumours. Thus FLIP may be implicated in tissue homeostasis as an important regulator of apoptosis.

Tags: Research Support, Non-U.S. Gov't

Descriptors: *Adaptor Proteins, Signal Transducing; *Apoptosis; *Carrier Proteins--physiology--PH; *Caspases; *Intracellular Signaling Peptides and Proteins; Amino Acid Sequence; Animals; Antigens, CD95--metabolism--ME; Carrier Proteins--genetics--GE; Carrier Proteins--

metabolism--ME; Cells, Cultured; Chromosomes, Human, Pair 2; Cloning, Molecular; Cysteine Endopeptidases--metabolism--ME; Humans; Lymphocyte Activation; Melanoma--metabolism--ME; Molecular Sequence Data; Sequence Homology ; Amino Acid; T-Lymphocytes--cytology--CY; T-Lymphocytes--immunology--IM; T-Lymphocytes--metabolism--ME; Tumor Cells, Cultured
Molecular Sequence Databank No.: GENBANK/U97074; GENBANK/U97075; GENBANK/U97076
CAS Registry No.: 0 (Adaptor Proteins, Signal Transducing); 0 (Antigens, CD95); 0 (CASP8 and FADD-like apoptosis regulating protein); 0 (Carrier Proteins); 0 (Intracellular Signaling Peptides and Proteins); 0 (MORT1 protein)
Enzyme No.: EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.22- (Caspases); EC 3.4.22- (caspase 8); EC 3.4.22- (caspase 9) Record Date Created: 19970724 Record Date Completed: 19970724

11/5/51 (Item 51 from file: 15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.
11928659 PMID: 9208847

Casper is a FADD - and caspase-related inducer of apoptosis .

Shu H B; Hsieh D R; Goeddel D V
Tularik, Incorporated, South San Francisco, California 94080, USA.
Immunity (UNITED STATES) Jun 1997, 6 (6) p751-63, ISSN 1074-7613, Journal Code: 9432918
Publishing Model Print Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM
Record type: MEDLINE; Completed Subfile: INDEX MEDICUS

Caspases are cysteine proteases that play a central role in apoptosis . Caspase - 8 may be the first enzyme of the proteolytic cascade activated by the Fas ligand and tumor necrosis factor (TNF). Caspase - 8 is recruited to Fas and TNF receptor-1 (TNF-R1) through interaction of its prodomain with the death effector domain (DED) of the receptor-associating FADD . Here we describe a novel 55 kDa protein, Casper, that has sequence similarity to caspase - 8 throughout its length. However, Casper is not a caspase since it lacks several conserved amino acids found in all caspases. Casper interacts with FADD , caspase - 8 , caspase - 3 , TRAF1 , and TRAF2 through distinct domains. When overexpressed in mammalian cells, Casper potently induces apoptosis . A C-terminal deletion mutant of Casper inhibits TNF- and Fas-induced cell death, suggesting that Casper is involved in these apoptotic pathways.

Descriptors: *Adaptor Proteins, Signal Transducing; *Antigens, CD95 --physiology--PH; * Apoptosis ; *Carrier Proteins--metabolism--ME; *Carrier Proteins--physiology--PH; *Caspases; *Cysteine Endopeptidases --chemistry--CH; *Intracellular Signaling Peptides and Proteins; *Tumor Necrosis Factor-alpha--physiology--PH; *Viral Proteins; Amino Acid Sequence ; Cloning, Molecular; Cysteine Endopeptidases--metabolism--ME; Endopeptidases--metabolism--ME; Enzyme Induction; HeLa Cells; Humans; Molecular Sequence Data; Protein Binding; Protein Processing, Post-Translational; Proteins--metabolism--ME; Recombinant Proteins; Sequence Alignment; Sequence Deletion; Sequence Homology , Amino Acid; Serpins--metabolism--ME; Signal Transduction; Structure-Activity Relationship; TNF Receptor-Associated Factor 1; TNF Receptor-Associated Factor 2

Molecular Sequence Databank No.: GENBANK/U97074

CAS Registry No.: 0 (Adaptor Proteins, Signal Transducing); 0 (Antigens, CD95); 0 (CASP8 and FADD-like apoptosis regulating protein); 0 (Carrier Proteins); 0 (Intracellular Signaling Peptides and Proteins); 0 (MORT1 protein); 0 (Proteins); 0 (Recombinant Proteins); 0 (Serpins); 0 (TNF Receptor-Associated Factor 1); 0 (TNF Receptor-Associated Factor 2); 0 (Tumor Necrosis Factor-alpha); 0 (Viral Proteins); 96282-35-8 (interleukin-1beta-converting enzyme inhibitor)
Enzyme No.: EC 3.4- (Endopeptidases); EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.22- (Caspases); EC 3.4.22- (caspase 8); EC 3.4.22- (caspase 9); EC 3.4.22- (caspase - 3) Record Date Created: 19970728 Record Date Completed: 19970728

11/7/65 (Item 65 from file: 15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.

11830217 PMID: 9087414

Viral FLICE: inhibitory proteins (FLIPs) prevent apoptosis induced by death receptors

Thome M; Schneider P; Hofmann K; Fickenscher H; Meiri E; Neipel F; Maltmann C; Burns K; Bodmer J L; Schrotter M; Scaffidi C; Kramer P H; Peter M E; Tschoop J

Institute of Biochemistry, University of Lausanne, Epalinges, Switzerland.

Nature (ENGLAND) Apr 3 1997, 386 (6624) p517-21, ISSN 0028-0836, Journal Code: 0410462

Publishing Model Print Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM

Record type: MEDLINE; Completed

12/6/1 (Item 1 from file: 15 1792216 PMID: 15928597

[FLIP--an enemy which might lose the battle against the specific inhibitors of translation] FLIP-- przeciwnik, który moze przegrac pojedynczy walec z czynnikiem swoiste hamujacymi translae.
Apr 11 2005

12/6/2 (Item 2 from file: 15 14974690 PMID: 13679070

Casper /c-FLIP is physically and functionally associated with NF-kappaB1 p105. Oct 3 2003

12/6/3 (Item 3 from file: 15 14010128 PMID: 11755527

The short splice form of Casper /c-FLIP is a major cellular inhibitor of TRAIL-induced apoptosis .
Jan 2 2002

12/6/4 (Item 4 from file: 15 12942169 PMID: 10894163

Requirement for Casper (c-FLIP) in regulation of death receptor-induced apoptosis and embryonic development. Jun 2000

12/6/5 (Item 5 from file: 15 12820361 PMID: 10753878

Activation of NF-kappaB by FADD, Casper , and caspase-8. Apr 14 2000

Viruses have evolved many distinct strategies to avoid the host's apoptotic response. Here we describe a new family of viral inhibitors (v-FLIPs) which interfere with apoptosis signalled through death receptors and which are present in several gamma-herpesviruses (including Kaposi's-sarcoma-associated human herpesvirus-8), as well as in the tumorigenic human molluscipoxvirus. v-FLIPs contain two death-effector domains which interact with the adaptor protein FADD , and this inhibits the recruitment and activation of the protease FLICE by the CD95 death receptor. Cells expressing v-FLIPs are protected against apoptosis induced by CD95 or by the related death receptors TRAMP and TRAIL-R. The herpesvirus saimiri FLIP is detected late during the lytic viral replication cycle, at a time when host cells are partially protected from CD95-ligand-mediated apoptosis . Protection of virus-infected cells against death-receptor-induced apoptosis may lead to higher virus production and contribute to the persistence and oncogenicity of several FLIP-encoding viruses. Record Date Created: 19970428 Record Date Completed: 19970428

11/7/110 (Item 110 from file: 15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.

1312002 PMID: 8643514

Cloning and expression of apoptosis inhibitory protein homologs that function to inhibit apoptosis and/or bind tumor necrosis factor receptor-associated factors.

Uren A G; Pakusch M; Hawkins C J; Puls K L; Vaux D L

The Walter and Eliza Hall Institute of Medical Research, Victoria, Australia.

Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) May 14 1996, 93 (10) p4974-8, ISSN 0027-8424, Journal Code: 7505876, Publishing Model Print Document type: Journal Article

Languages: ENGLISH Main Citation Owner: NLM Record type: MEDLINE; Completed

Baculovirus inhibitors of apoptosis (IAPs) act in insect cells to prevent cell death. Here we describe three mammalian homologs of IAP, MIHA, MIHB, and MIHC, and a Drosophila IAP homolog , DIHA. Each protein bears three baculovirus IAP repeats and an N-terminal ring finger motif. Apoptosis mediated by interleukin 1beta converting enzyme (ICE), which can be inhibited by Orgyia pseudotsugata nuclear polyhedrosis virus IAP (OpiAP) and coxopox virus cmaA, was also inhibited by MIHA and MIHB. As MIHB and MIHC were able to bind to the tumor necrosis factor receptor-associated factors TRAF1 and TRAF2 in yeast two-hybrid assays, these results suggest that IAP proteins that inhibit apoptosis may do so by regulating signals required for activation of ICE-like proteases. Record Date Created: 19960718 Record Date Completed: 19960718

11/7/115 (Item 115 from file: 15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.

11234430 PMID: 8548810

The TNFR2-TRAF signaling complex contains two novel proteins related to baculoviral inhibitor of apoptosis proteins.

Rothe M; Pan M G; Henzel W J; Ayres T M; Goeddel D V

Department of Molecular Biology Tularik, Incorporated, South San Francisco, California 94080, USA.

Cell (UNITED STATES) Dec 29 1995, 83 (7) p1243-52, ISSN 0092-8674, Journal Code: 0413066

Publishing Model Print Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM

Record type: MEDLINE; Completed

The 75 kDa tumor necrosis factor receptor (TNFR2) transduces extracellular signals via receptor-associated cytoplasmic proteins. Two of these signal transducers, TRAF1 and TRAF2, were isolated and characterized previously. We report here the biochemical purification and subsequent molecular cloning of two novel TNFR2-associated proteins, designated c-IAP1 and c-IAP2, that are closely related mammalian members of the inhibitor of apoptosis protein (IAP) family originally identified in baculoviruses. The viral and cellular IAPs contain N-terminal baculovirus IAP repeat (BIR) motifs and a C-terminal RING finger. The c-IAPs do not directly contact TNFR2, but rather associate with TRAF1 and TRAF2 through their N-terminal BIR motif comprising domain. The recruitment of c-IAP1 or c-IAP2 to the TNFR2 signaling complex requires a TRAF2 - TRAF1 heterocomplex. Record Date Created: 19960221 Record Date Completed: 19960221

12/6/11 (Item 3 from file: 0013647738 BIOSIS NO.: 200200241249

Cellular adhesion inhibits CD95/Fas-mediated apoptosis by altering the intracellular localization and availability of c-FLIP. 2001

12/6/12 (Item 4 from file: 0013497049 BIOSIS NO.: 200200090560

The short splice form of Casper /c-FLIP is a major cellular inhibitor of TRAIL-induced apoptosis 2002

12/6/13 (Item 5 from file: 0013377655 BIOSIS NO.: 200100549494

Regulators of apoptosis 2001

12/6/14 (Item 6 from file: 0012734815 BIOSIS NO.: 200000453128

Activation of NF-kappaB by FADD, Casper , and caspase-8 2000

12/6/15 (Item 7 from file: 0012616285 BIOSIS NO.: 200000334598

Requirement for casper (c-FLIP) in regulation of death receptor-induced apoptosis and embryonic development 2000

12/6/16 (Item 8 from file: 0011461171 BIOSIS NO.: 199800255418
Cell death attenuation by 'Usurpin', a mammalian DED-caspase homologue that precludes caspase-8 recruitment and activation by the CD-95 (Fas, APO-1) receptor complex. 1998

12/5/12 (Item 2 from file: 15 DIALOG(R)File 155: MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.
14974690 PMID: 13679070
Casper /c-FLIP is physically and functionally associated with NF-kappaB1 p105.
Li Zhiqin; Zhang Jingbo; Chen Danyang; Shu Hong-Bing
Department of Cell Biology, Institute of Basic Medical Sciences, Chinese Academy of Medical Sciences, Beijing 10005, China.
Biochemical and biophysical research communications (United States) Oct 3 2003, 309 (4) p980-5, ISSN 0006-291X. Journal Code: 0372516 Publishing Model Print. Document type: Journal Article Languages: ENGLISH
Main Citation Owner: NLM Record type: MEDLINE; Completed Subfile: INDEX MEDICUS
Casper /c-FLIP is a caspase-8-related molecule critically involved in regulation of death receptor-induced apoptosis. It has been shown that Casper can either promote or antagonize apoptosis and can activate the transcription factor NF-kappaB. The exact functions of Casper are controversial. To further understand how Casper signals, we searched Casper-interacting proteins by yeast two-hybrid screening. This effort identified NF-kappaB1 (p105), an atypical IkkappaB molecule and the precursor of NF-kappaB subunit p50. Co-immunoprecipitation experiments indicated that Casper interacted with p105 in 293 cells and this interaction was mediated through the C-terminal IkkappaB-like domain (IkkappaBgamma). Overexpression of p105 and IkkappaBgamma inhibited Casper-induced NF-kappaB activation and potentiated Casper-induced apoptosis. Furthermore, Casper and its C-terminal caspase-like domain inhibited p105 processing into p50. Our findings suggest that p105 is involved in Casper-mediated regulation of apoptosis and NF-kappaB activation.
Tags: Research Support, Non-U.S. Govt
Descriptors: *Carrier Proteins-physiology-PH; *Intracellular Signaling Peptides and Proteins; *NF-kappa B-metabolism-ME; *Protein Precursors-metabolism-ME; Carrier Proteins-metabolism-ME; Cell Line; Humans; Protein Binding; Two-Hybrid System Techniques
CAS Registry No.: 0 (Casper) and FADD-like apoptosis regulating protein); 0 (Carrier Proteins); 0 (Intracellular Signaling Peptides and Proteins); 0 (NF-kappa B); 0 (NF-kappa B p105 precursor); 0 (Protein Precursors) Record Date Created: 20030918 Record Date Completed: 20031204

12/5/4 (Item 4 from file: 15 DIALOG(R)File 155: MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.
12942169 PMID: 10894163
Requirement for Casper (c-FLIP) in regulation of death receptor-induced apoptosis and embryonic development.
Yeh W C; Iie A; Elia A J; Ng M; Shu H B; Wakeham A; Mirsós C; Suzuki N; Bonnard M; Goeddel D V; Mak T W
Angen Institute, Department of Medical Biophysics, University of Toronto and Ontario Cancer Institute, Canada.
Immunology (UNITED STATES) Jun 2000, 12 (6) p633-42, ISSN 1074-7613 Journal Code: 9432918
Publishing Model Print. Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM
Record type: MEDLINE; Completed Subfile: INDEX MEDICUS
Casper (c-FLIP) associates with FADD and caspase-8 in signaling complexes downstream of death receptors like Fas. We generated Casper-deficient mice and cells and noted a duality in the physiological functions of this molecule. casper -/- embryos do not survive past day 10.5 of embryogenesis and exhibit impaired heart development. This phenotype is reminiscent of that reported for FADD-/- and caspase-8-/- embryos. However, unlike FADD-/- and caspase-8-/- cells, casper -/- embryonic fibroblasts are highly sensitive to FasL- or TNF-induced apoptosis and show rapid induction of caspase activities. NF-kappaB and JNK/SAPK activation is intact in TNF-stimulated casper -/- cells. These results suggest that Casper has two distinct roles: to cooperate with FADD and caspase-8 during embryonic development and to mediate cytoprotection against death factor-induced apoptosis.
Tags: Female; Mice; Research Support, Non-U.S. Govt
Descriptors: *Apoptosis-immunology-IM; *Carrier Proteins-physiology-PH; *Embryonic and Fetal Development-immunology-IM; *Intracellular Signaling Peptides and Proteins; *Receptors, Tumor Necrosis Factor-physiology-PH; Animals; Apoptosis-genetics-GE; Carrier Proteins-genetics-GE; Caspases-metabolism-ME; Cell Line; Embryonic and Fetal Development-genetics-GE; Enzyme Activation-immunology-IM; Heart-embryology-EM; JNK Mitogen-Activated Protein Kinases; Mice; Mice, Inbred C57BL; Mice, Knockout; Mitogen-Activated Protein Kinases-metabolism-ME; Myocardium-enzymology-EN; Myocardium-immunology-IM; Myocardium-metabolism-ME; Myocardium-pathology-PA; NF-kappa B-metabolism-ME; Stem Cells-enzymology-EN; Stem Cells-immunology-IM; Stem Cells-metabolism-ME; Stem Cells-pathology-PA; Tumor Necrosis Factor-alpha-physiology-PH
CAS Registry No.: 0 (CASP8 and FADD-like apoptosis regulating protein); 0 (Carrier Proteins); 0 (Intracellular Signaling Peptides and Proteins); 0 (NF-kappa B); 0 (Receptors, Tumor Necrosis Factor); 0 (Tumor Necrosis Factor-alpha)
Enzyme No.: EC 2.7.1.37 (JNK Mitogen-Activated Protein Kinases); EC 2.7.1.37 (Mitogen-Activated Protein Kinases); EC 3.4.22.1 (caspase 9); EC 3.4.22.1 (caspase-3) Record Date Created: 20000731 Record Date Completed: 20000731
15/6/1 (Item 1 from file: 15 DIALOG(R)File 155: MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.
156/1 (Item 1 from file: 15 DIALOG(R)File 155: MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.
Differential modulation of apoptosis sensitivity in CD95 type I and type II cells. Aug 6 1999
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14974690 PMID: 13679070
The role of c-FLIP in modulation of CD95-induced apoptosis. Jan 15 1999

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14974690 PMID: 13679070
Expression of c-FLIP(L) and resistance to CD95-mediated apoptosis of monocyte-derived dendritic cells: inhibition by bisindolylmaleimide. Jun 1 2000

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Caspase activation is required for nitric oxide-mediated, CD95(APO-1/Fas)-dependent and independent apoptosis in human neoplastic lymphoid cells. Jun 1 1998

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14974690 PMID: 13679070
Two CD95 (APO-1/Fas) signaling pathways. Mar 16 1998

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Bcl-xL acts downstream of caspase-8 activation by the CD95 death-inducing signaling complex. Feb 6 1998

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14974690 PMID: 13679070
Caspase activation is required for nitric oxide-mediated, CD95(APO-1/Fas)-dependent and independent apoptosis in human neoplastic lymphoid cells. Jun 1 1998

15/6/8 (Item 8 from file: 15 DIALOG(R)File 155: MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.
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Bcl-xL acts downstream of caspase-8 activation by the CD95 death-inducing signaling complex. Feb 6 1998

12/5/5 (Item 5 from file: 15 DIALOG(R)File 155: MEDLINE(R) (c) format only 2005 Dialog. All rts. reserv.
12820361 PMID: 10753878
Activation of NF-kappaB by FADD, Casper, and caspase-8.
Hu W H; Johnson H; Shu H B
Department of Immunology, National Jewish Medical and Research Center, University of Colorado Health Sciences Center, Denver, Colorado 80206, USA.
Journal of biological chemistry (UNITED STATES) Apr 14 2000, 275 (1) p10838-44, ISSN 0021-9258. Journal Code: 2985121R
Publishing Model Print. Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM
Record type: MEDLINE; Completed Subfile: INDEX MEDICUS
Fas-associated death domain protein (FADD), caspase-8-related protein (Casper), and caspase-8 are components of the tumor necrosis factor receptor type 1 (TNF-R1) and Fas signaling complexes that are involved in TNF-R1- and Fas-induced apoptosis. Here we show that overexpression of FADD and Casper potentially activates NF-kappaB. In the presence of caspase inhibitors, overexpression of caspase-8 also activates NF-kappaB. A caspase-inactive point mutant, caspase-8(C360S), activates NF-kappaB as potently as wild-type caspase-8, suggesting that caspase-8-induced apoptosis and NF-kappaB activation are uncoupled. NF-kappaB activation by FADD and Casper is inhibited by the caspase-specific inhibitors crmA and BD-fmk, suggesting that FADD- and Casper-induced NF-kappaB activation is mediated by caspase-8. FADD, Casper, and caspase-8-induced NF-kappaB activation are inhibited by dominant negative mutants of TRAF2, NIK, IkkappaB kinase alpha, and IkkappaB kinase beta. A dominant negative mutant of RIP inhibits FADD- and caspase-8-induced but not Casper-induced NF-kappaB activation. A mutant of Casper and the caspase-specific inhibitors crmA and BD-fmk partially inhibit TNF-R1-, TRADD, and TNF-induced NF-kappaB activation, suggesting that FADD, Casper, and caspase-8 function downstream of TRADD and contribute to TNF-R1-induced NF-kappaB activation. Moreover, activation of caspase-8 results in proteolytic processing of NIK, which is inhibited by crmA. When overexpressed, the processed fragments of NIK do not activate NF-kappaB, and the processed C-terminal fragment inhibits TNF-R1-induced NF-kappaB activation. These data indicate that FADD, Casper, and pro-caspase-8 are parts of the TNF-R1-induced NF-kappaB activation pathways, whereas activated caspase-8 can negatively regulate TNF-R1-induced NF-kappaB activation by proteolytically inactivating NIK.
Tags: Research Support, Non-U.S. Govt
Descriptors: *Arabidopsis Proteins; *Carrier Proteins-physiology-PH; *Caspases-physiology-PH; *Fatty Acid Desaturases-physiology-PH; *Intracellular Signaling Peptides and Proteins; *NF-kappa B-metabolism-ME; Antigen; CD-physiology-PH; Apoptosis; Caspases-antagonists and inhibitors-AI; Humans; Protein-Serine-Threonine Kinases-physiology-PH; Receptors, Tumor Necrosis Factor-physiology-PH; Receptors, Tumor Necrosis Factor, Type I
CAS Registry No.: 0 (Antigens, CD); 0 (Arabidopsis Proteins); 0 (CASP8 and FADD-like apoptosis regulating protein); 0 (Carrier Proteins); 0 (Intracellular Signaling Peptides and Proteins); 0 (NF-kappa B); 0 (Receptors, Tumor Necrosis Factor); 0 (Receptors, Tumor Necrosis Factor, Type I)
Enzyme No.: EC 1.14.99.- (Fad7 protein, Arabidopsis); EC 1.14.99.- (Fatty Acid Desaturases); EC 2.7.1.- (NF-kappa B kinase); EC 2.7.1.37 (Protein-Serine-Threonine Kinases); EC 3.4.22.- (Caspases); EC 3.4.22.- (caspase 9); EC 3.4.22.- (caspase 9) Record Date Created: 20000505 Record Date Completed: 20000505

12/7/13 (Item 5 from file: DIALOG(R)File 5: Biosis Previews(R) (c) 2005 BIOSIS. All rts. reserv.
0013377655 BIOSIS NO.: 200100549494
Regulators of apoptosis
AUTHOR: Shu Hong-Bing (Reprint); Goeddel David V
AUTHOR ADDRESS: South San Francisco, CA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1247 (1). June 5, 2001 2001
MEDIUM: e-file ISSN: 0098-1133 DOCUMENT TYPE: Patent RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The invention provides methods and compositions relating to apoptosis regulating proteins, known as Casper proteins, and related nucleic acids. The proteins may be produced recombinantly from transformed host cells from the disclosed Casper encoding nucleic acid or purified from human cells. The invention provides specific hybridization probes and primers capable of specifically hybridizing with the disclosed Casper gene, Casper-specific binding agents such as specific antibodies, and methods of making and using the subject compositions in diagnosis, therapy and in the biopharmaceutical industry.

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- 15/6/11 (Item 11 from file: 15 11905712 PMID: 9184224)
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